resins using HPLC with the fluorescence detector [abstract; in Japanese]. Jpn J Dent Mater 17(special issue 32):119 (1998).
4. Schmalz G, Preiss A, Arenholt-Bindslev D. Bisphenol A content of resin monomers and degradation products[abstract]. J Dent Res 77(special issue B):823 (1998).
5. Nathanson D, Lertpitayakun P, Lamkin MS, Mahnaz EB, Lee-Chou L. In vitro elution of leachable components from dental sealants. J Am Dent Assoc 128:1517–1523 (1997).
6. Steinmetz R, Brown NG, Allen DL, Bigsby RM, Ben-Jonathan N. The environmental estrogen bisphenol A stimulates prolactin release in vitro and in vivo. Endocrinology 138:1780–1786 (1997).
7. Colerangle JB, Deodutta R. Profound effects of the weak environmental estrogen-like chemical bisphenol A on the growth of the mammary gland of Noble rats. J Steroid Biochem Mol Biol 60:153–160 (1997).
8. Pfeiffer E, Rosenberg B, Deuschel S, Metzler M. Interference with microtubules and induction of micronuclei in vitro by various bisphenols. Mutat Res 390:21–31 (1997).
9. Dodge JA, Glasebrook AL, Magee DE, Phillips DL, Sato M, Short LL, Bryant HU. Enviromental estrogens: effects on cholesterol lowering and bone in the ovariectomized rat. J Steroid Biochem Mol Biol 59:155–161 (1996).
10. Ratnasabapathy R, Tom M, Post C. Modulation of the hepatic expression of the estrogen-regulated messenger-RNA stabilizing factor by estrogenic and antiestrogenic nonsteroidal xenobiotics. Biochem Pharmacol 53 :1425–1434 (1997).
11. Nagel SC, vom Saal FS, Thayer KA, Dhar MG, Boechler M, Welshons WV. Relative binding affinity-serum modified access (RBA-SMA) assay predicts the relative *in vivo* bioactivity of the xenoestrogens bisphenol A and octylphenol. Environ Health Perspect 105:70–76 (1997).
12. Pérez P, Pulgar R, Olea-Serrano F, Villalobos M, Rivas A, Metzler M, Pedraza V, Olea N. The estrogenicity of bisphenol A-related diphenylalkanes with various substituents at the central carbon and the hydroxy groups. Environ Health Perspect 106:167–174 (1998).
13. Soderholm KJ, Mariotti A. BIS-GMA–based resins in dentistry: are they safe? J Am Dent Assoc 130:201–209 (1999).

Error in DEHP Background Concentration

In the May 1998 issue of *Environmental Health Perspectives,* Woodruff et al. (*1*) reported an analysis conducted as part of the U.S. Environmental Protection Agency (EPA) Cumulative Exposure Project (CEP). The EPA modeled air concentrations of chemicals listed in the Clean Air Act as hazardous air pollutants (HAPs) in over 60,000 census tracts and compared those concentrations to health benchmarks. The Phthalate Esters Panel of the Chemical Manufacturers Association has become aware of an error in the background concentration value used for bis(2-ethylhexyl) phthalate (DEHP), with the result that modeled air concentrations for DEHP—and thus the potential health hazard—were greatly exaggerated.

Woodruff et al. (*1*) reported, "Eight pollutants ... [including DEHP] had modeled concentrations exceeding the benchmark concentrations for cancer in 100% of the census tracts. For each of these HAPs, the background concentration alone ... exceeded the benchmark concentration for cancer, as shown in Table 2." Their Table 2 (*1*) shows that the background concentration used for DEHP was 1.6 $\mu g/m^3$. That value was taken from Howard (*2*) who reported "mean remote ocean air concentrations" for DEHP of 0.07–0.17 ppb, citing Atlas and Giam (*3*). However, Atlas and Giam (*3*) actually reported remote DEHP air concentrations to be 0.32–2.68 ng/m^3, with a mean of 1.4 ng/m^3—a value more than 1,000 times less than the background value used for the CEP analysis. The panel has alerted the EPA to this error, and the EPA accordingly has corrected the CEP modeling report (*4*).

Table 2 of Woodruff et al. (*1*) shows that if the erroneous background value of 1.6 $\mu g/m^3$ is disregarded, the CEP model predicts DEHP air concentrations to exceed the health benchmark of 0.25 $\mu g/m^3$ in only 18 census tracts. Even this estimate probably exaggerates the potential health hazard for two reasons. First, to the panel's knowledge, the highest measured ambient DEHP air concentration in the United States that has been reported in the literature is 28 ng/m^3 (*5*)—an order of magnitude below the EPA's cancer health benchmark. Second, the EPA's health benchmark of 0.25 $\mu g/m^3$ was derived using an upper-bound unit risk methodology to extrapolate tumor data in rats and mice to human risk (*6,7*). However, numerous investigators now conclude that peroxisome proliferators such as DEHP pose little if any human cancer risk and that the quantitative risk assessment for such compounds should be based on a margin of exposure approach (*8–10*). This would significantly increase the health benchmark for DEHP and decrease (probably to zero) the number of census tracts in which modeled air concentrations would exceed the health benchmark.

Courtney M. Price
CHEMSTAR
Arlington, Virginia

References and Notes

1. Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. Environ Health Perspect 106:245–251 (1998).
2. Howard P. Di(2-ethylhexyl) phthalate. In: Handbook of Environmental Fate and Exposure Data for Organic Chemicals. Vol. I: Large Production and Priority Pollutants. Chelsea, MI:Lewis Publishers, 1989.
3. Atlas E, Giam CS. Global transport of organic pollutants: ambient concentrations in the remote marine atmosphere. Science 211:163–165 (1981).
4. Rosenbaum A, Ligocki M, Wei Y. Modeling Cumulative Outdoor Concentrations of Hazardous Air Pollutants. San Rafael, CA:ICF Kaiser, Systems Applications International Division, 1998. Available: http://www.epa.gov/CumulativeExposure/resource/report.htm [updated 8 December 1998].
5. Sheldon L, Whitaker D, Keever J, Clayton A, Perritt R. Phthalates and PAHs in indoor and outdoor air in a southern California community. Proc Indoor Air 3:109-114 (1993).
6. Caldwell JC, Woodruff TJ, Morello-Frosch R, Axelrad DA. Application of health information to hazardous air pollutants modeled in EPA's Cumulative Exposure Project. Toxicol Ind Health 14:429–454 (1998).
7. US EPA. Integrated Risk Information System Database for di(2-ethylhexyl)phthalate (DEHP), CASRN 117-81-7. Available: http://www.epa.gov/ngispgm3/iris/subst/0014.htm [updated 19 May 1998].
8. Cattley RC, DeLuca J, Elcombe C, Fenner-Crisp P, Lake BG, Marsman DS, Pastoor TA, Popp JA, Robinson DE, Schwetz B, et al. Do peroxisome proliferating compounds pose a hepatocarcinogenic hazard to humans? Regul Toxicol Pharmacol 27:47–60. (1998).
9. Huber WW, Grasl-Kraupp B, Schulte-Herman R. Hepatocarcinogenic potential of di(2-ethylhexyl) phthalate in rodents and its implications on human risk. Crit Rev Toxicol 26(4):365–481 (1996).
10. Lake BG. Mechanisms of hepatocarcinogenicity of peroxisome-proliferating drugs and chemicals. Ann Rev Pharmacol Toxicol 35:483–507 (1995).

DEHP Correction

We thank Courtney Price for pointing out an error in the background concentration for bis(2-ethylhexyl)phthalate (DEHP) in our paper "Public Health Implications of 1990 Air Toxics Concentrations across the United States" (*1*). In this paper, emissions data from stationary and mobile sources are used in an atmospheric dispersion model to estimate outdoor concentrations of 148 toxic air contaminants for each of the 60,803 census tracts in the contiguous United States. Outdoor concentrations of air toxics were compared to previously defined benchmark concentrations for cancer and noncancer health effects. Benchmark concentrations are based on standard toxicological references and represent air toxic levels above which health risks may occur.

The results reported for DEHP are incorrect due to an error in the estimated background concentration for DEHP. We had originally used a value of 1.6 $\mu g/m^3$ for DEHP, which was reported by Howard (*2*). As pointed out by Price, Howard (*2*) had incorrectly reported the value from another source, Atlas and Giam (*3*). Consequently, we have revised the background concentration for DEHP to 0.0014 $\mu g/m^3$, consistent with the mean value reported by Atlas and Giam.

We had reported that the background concentration for DEHP was greater than the cancer benchmark for DEHP. However, the revised background concentration is much lower than the cancer benchmark. Thus, DEHP should not be included in the list of pollutants in Table 2 for which background concentrations alone exceeded cancer benchmark concentrations (*1*). The

number of benchmark concentrations exceeded by modeled concentrations now ranges from 7 to 31 per census tract, with a mean of 13; approximately half the census tracts have between 10 and 14 estimated hazardous air pollutant concentrations that exceeded benchmark concentrations.

Price also questions whether any concentrations of DEHP would be high enough to exceed the cancer benchmark. The estimated concentrations for DEHP were based primarily on emissions from the Toxics Release Inventory, which relies on self-reporting of estimated emissions from the industry to the public.

We appreciate input on the analysis. In addition, the U.S. EPA Office of Air Quality Planning and Standards is planning to update the modeled air toxics concentration estimates with emissions data for 1996. Continued feedback on the inputs to the model will help improve the concentration estimates.

Tracey Woodruff
U.S. Environmental Protection Agency
Washington, DC

References and Notes

1. Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. Environ Health Perspect 106:245–251 (1998).
2. Howard P. Handbook of Fate and Exposure Data for Organic Chemicals. Vol I: Large Production and Priority Pollutants. Chelsea, MI:Lewis Publishers, 1989.
3. Atlas E, Giam C. Global transport of organic pollutants: ambient concentrations in the remote marine atmosphere. Science 211:163–165 (1981).

Corrections and Clarifications

The photograph by David Tenenbaum used to accompany the article "Fertilizing or Contaminating?" [*EHP* 107(3):A137 (1999)] shows the application of regulated sewage sludge by a municipal sewerage district, not the application of industrial sludge as the caption implies. *EHP* regrets any confusion caused by the use of this photo.